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PERIOPERATIVE AND CRITICAL CARE VENTILATION (L BALL, SECTION EDITOR)

Perioperative Ventilation in Neurosurgical Patients: Considerations and Challenges

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Abstract

Purpose of Review The aim of this narrative review is to summarize critical considerations for perioperative airway management and mechanical ventilation in patients undergoing neurosurgical procedures.

Recent Findings Given the significant influence that ventilation has on intracranial pressure (ICP) and cerebral blood flow, ventilator settings need to be carefully managed. For example, high positive end-expiratory pressure (PEEP) can increase ICP, while hyperventilation can reduce it. Finding the optimal balance is the key. While evidence supporting lung-protective ventilation in neurosurgical patients is limited, preliminary data suggest that its use could be beneficial, similar to general surgical patients. This typically involves using lower tidal volumes and maintaining optimal oxygenation to prevent ventilator-associated lung injury. Airway management in neurosurgical patients must consider the risk of increased ICP during intubation and the potential for airway complications. Techniques like rapid sequence induction and the use of neuromuscular blockers may be employed to minimize these risks. The primary goal of ventilation in neurosurgical patients is to maintain adequate oxygenation and carbon dioxide removal while minimizing harm to the lungs and brain. However, there may be exceptions where specific ventilatory adjustments are needed, such as in cases of compromised gas exchange or elevated ICP.

Summary Patients undergoing neurosurgical procedures often require invasive ventilation due to the complexities of the operation and the need to manage the airway. This creates unique challenges because ventilator settings must balance the need to protect both the lungs and the brain. Further research is needed to establish clear guidelines and optimize ventilatory care in this population.

Keywords Airway management · Mechanical ventilation · Neurosurgical procedures · Brain-lung crosstalk

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Abbreviations

ABI	Acute brain injury
ABP	Arterial blood pressure
ARDS	Aacute respiratory distress syndrome
CA	Cerebral autoregulation
CBF	Cerebral blood flow
$CMRO_2$	Cerebral metabolic rate of oxygen
CPP	Cerebral perfusion pressure
CSF	Cerebrospinal fluid
ΔP	Driving Pressure
EEG	Electroencephalogram
$ETCO_2$	End tidal CO_2
FiO_2	Fraction inspirated of oxygen
HFNC	High flow nasal cannula
ICP	Intracranial pressure
ICU	Intensive care unit
LPV	Lung-protective ventilation
MAP	Median artery pressure
MV	Mechanical ventilation
MP	Mechanical power
OSAS	Obstructive sleep apnea syndrome
PBW	Predicted body weight
PEEP	Positive end-expiratory pressure
PPC	Postoperative pulmonary complications
Pplat	Plateau Pressure
RCT	Randomized controlled trial
RMs	Recruitment maneuvers
ROS	Reactive oxygen species
RSI	Rapid sequence intubation
rSO_2	Regional cerebral saturation of oxygen
TBI	Traumatic brain injured
VAE	Venous air embolism
VILI	Ventilator-associated lung injury
V_T	Tidal volume

Introduction

In acute brain injury (ABI), patients undergoing neurosurgical procedures often require sedation and invasive mechanical ventilation (MV) for airway protection and ventilatory support. However, invasive MV poses potential risks to several organ systems and, therefore, necessitates careful considerations when choosing ventilatory strategies. A balance between preventing both ventilator-induced lung injury (VILI) and brain injury requires careful consideration, with the latter requiring monitoring of hemodynamics, cerebral perfusion and intracranial pressure (ICP) [1].

Little is known about MV in neurosurgical patients and most of the data stem from perioperative surgical or intensive care unit (ICU) cohorts. Lung-protective ventilation (LPV), a strategy discussed in the MV section, is employed during various surgical procedures [2] to prevent postoperative pulmonary complications (PPCs) [3] such as VILI [4]; however, it is often avoided in patients undergoing neurosurgical anesthesia [5].

Another critical consideration is the bi-directional *brain-lung crosstalk* [6–8], which may increase the risk of secondary brain injury [9]. *Brain-lung crosstalk* is mediated by multiple mechanisms [10]. After brain injury, inflammatory responses, hormonal dysregulation and catecholamine surges, result in increased expression of injurious molecular patterns and affect the lymphatic system [11]. This immune activation can directly damage type II pneumocytes, increasing pulmonary vascular hydrostatic pressure and lung capillary permeability, ultimately leading to lung injury [6, 8, 12–14].

Given the challenges associated with neurosurgical procedures, this review summarizes the current knowledge on specific airway management, MV parameters, thresholds to avoid brain and lung injury, extubation, and the risk of PPCs in patients undergoing neurosurgical procedures.

Specific Airway Management Considerations in Neurosurgical Patients

Positioning, cervical spine stability, hemodynamics during induction and the effects of anesthesia on ICP are unique conditions that may complicate airway management in neurosurgical patients [15]. Neurosurgical approaches involving the pituitary gland, temporal lobe or occipital-cervical zone, such as the Halo position or stereotactic head frame, often require forced unnatural head positions, leading to difficult airway management or failed intubation. Prolonged unnatural head positioning can impair cerebral venous outflow, potentially causing intraoperative brain swelling, increased ICP, ischemia, and cerebral infarction [15, 16]. The risk of ischemia is augmented in patients with large vessel occlusions [17].

Acute cervical spine injuries require immobilization and a cervical collar, hindering head extension during intubation, which may complicate airway management.

In addition, fluctuations in arterial blood pressure (ABP) can affect cerebral autoregulation, leading to poor postoperative outcomes like stroke, acute kidney injury, and delirium. It is important to individualize ABP targets to preserve cerebral perfusion [18]. Recent research, conducted in patients undergoing elective neurosurgery, monitored cerebral autoregulation (CA) using the correlation between changes in mean artery pressure (MAP) and regional cerebral O_2 saturation (rSO2). Although preoperative ABP value is often the target used to adjust the intraoperative ABP value, the optimal ABP (defined as the value above lower limit of CA)

is lower than the ABP at the baseline. These data may suggest the need of monitoring CA intraoperatively in order to personalized ABP target preserving cerebral perfusion [18].

Prolonged ventilation due to difficulties in airway management may lead to hypercapnia, hypoxemia and increased cerebral blood flow (CBF) with the risk of intracranial hypertension. In addition, laryngoscopy and intubation maneuvers often result in increase in systolic blood pressure by a mean of 20 mmHg [19], and consequentially in a rise of MAP and possibly of ICP especially in patients with impaired CA [20]. During this phase it is important to balance and maintain cerebral hemodynamics parameters as CBF, cerebral metabolic rate of oxygen ($CMRO_2$) and cerebrospinal fluid (CSF) dynamics by ensuring deep sedation, avoiding increased ICP and cerebral perfusion pressure (CPP) as consequence of extreme hyper- and hypotension, and the safeguarding of cerebral and spinal perfusion [21].

The optimal airway management strategy in neurosurgical patients is not yet defined and should be individualized. Generally, a focused medical history on potential difficult airway or on signs and symptoms of intracranial vascular insufficiency followed by a detailed physical examination (neck mobility, cervical cord stability etc.) and by the consultation of imaging is recommended in the preoperative period to anticipate and address eventual difficulties. In case of elective cranio-facial procedures and suspected difficult airway, a preoperative endoscopic airway examination could be considered in order to reduce the use of unnecessary awake intubation [22]. For patients with cervical spine instability, techniques minimizing head movement, maintaining head neutral position [23] with a manual in-line stabilization [24], are aimed to prevent secondary neurologic injury. A review suggests that conventional direct laryngoscopy may be inappropriate for securing the upper airway in cervical spine surgery [23]. Awake flexible fiberoptic intubation, nasal intubation, video-laryngoscopy and neck immobilization could be considered in case of unstable cervical spine patients [25]. If intraoperative intubation is necessary during awake surgery, a video-laryngoscope approach is indicated [21].

During endotracheal intubation, it is crucial to avoid hypoxia by ensuring adequate preoxygenation. Three minutes of preoxygenation with 100% of fraction inspirated of oxygen (FiO_2) or a short period of high flow nasal cannula (HFNC) is highly recommended [26]. The risk of hypercapnia and hypertension can be mitigated by using an appropriate level of sedation to blunt the sympathetic response to intubation [21]. In addition, care must be taken to prevent neck rotation which can lead to jugular vein compression and increases in intrathoracic pressure and ICP [27]. Weintraub et al. suggest that a prolonged (> 12 min) neck hyperextension could predispose to perioperative stroke because of transient changes in blood flow [28]. In a swine model, rapid sequence intubation (RSI) with paralysis seems to generate three-fold increases in peak of ICP compared with sedation only [29].

Furthermore, as recommended by American Society of Anesthesiologists guidelines during the intubation phase it is fundamental to use End tidal carbon dioxide ($ETCO_2$) monitoring as a standard of anesthetic monitoring to ensure adequate ventilation throughout the surgical procedure. This is extremely useful in neurosurgical patients, also because of the linear relation between $PaCO_2$ and cerebral blood flow. Supporting this, in cervical spine surgery patients with change of position from supine to prone, a recent study reported that $PaCO_2$ can decrease with no significant change in arterial to $ETCO_2$ value, suggesting that $ETCO_2$ can be used as a reliable guide to estimate $PaCO_2$ also during cervical spine procedures in prone position [30].

In any case, the best strategy for airway management seems to be the one with the highest possibility of success on the first attempt considering the operator's knowledge and with the lowest influence on patients [24]. Figure 1 summarizes issues in airway management in neurosurgical patients.

Mechanical Ventilation Strategies

LPV uses low tidal volumes (V_T) (6–8 ml/kg of PBW) and moderate positive end-expiratory pressure (PEEP) [31] Although LPV has been shown to reduce PPCs in other surgical contexts [3, 32–34], its use in neurosurgical patients is controversial.

In this line, the use of low V_T during LPV can result in hypercapnia with consequent harmful effects, such as cerebral vasodilatation and raised ICP [35–37]. In addition, PEEP may increase intrathoracic pressure, with a potential risk of intracranial hypertension due to diminished outflow from the jugular veins [7]. For the same reasons, a future study of permissive hypercapnia, named 'PERMISS' will exclude neurocritical care patients, explicitly.

Optimal MV parameters in neurosurgical anesthesia remains to be defined [38]. A post-hoc analysis of 'Local Assessment of Ventilatory management during general anesthesia for surgery' (LAS VEGAS) showed that neurosurgical patients were commonly ventilated with low V_T (median V_T 8 ml/kg PBW (IQR = 7.3– 9) and low PEEP (median PEEP = 5 cmH_2O (IQR 3–5), while RMs were rarely applied (6.9% of routine RMs vs. 1.4% of unplanned RMs) [39]. In this analysis, a similar incidence in PPCs (unplanned need for oxygen therapy, respiratory failure, ARDS, pneumonia, pneumothorax) was found in both spine

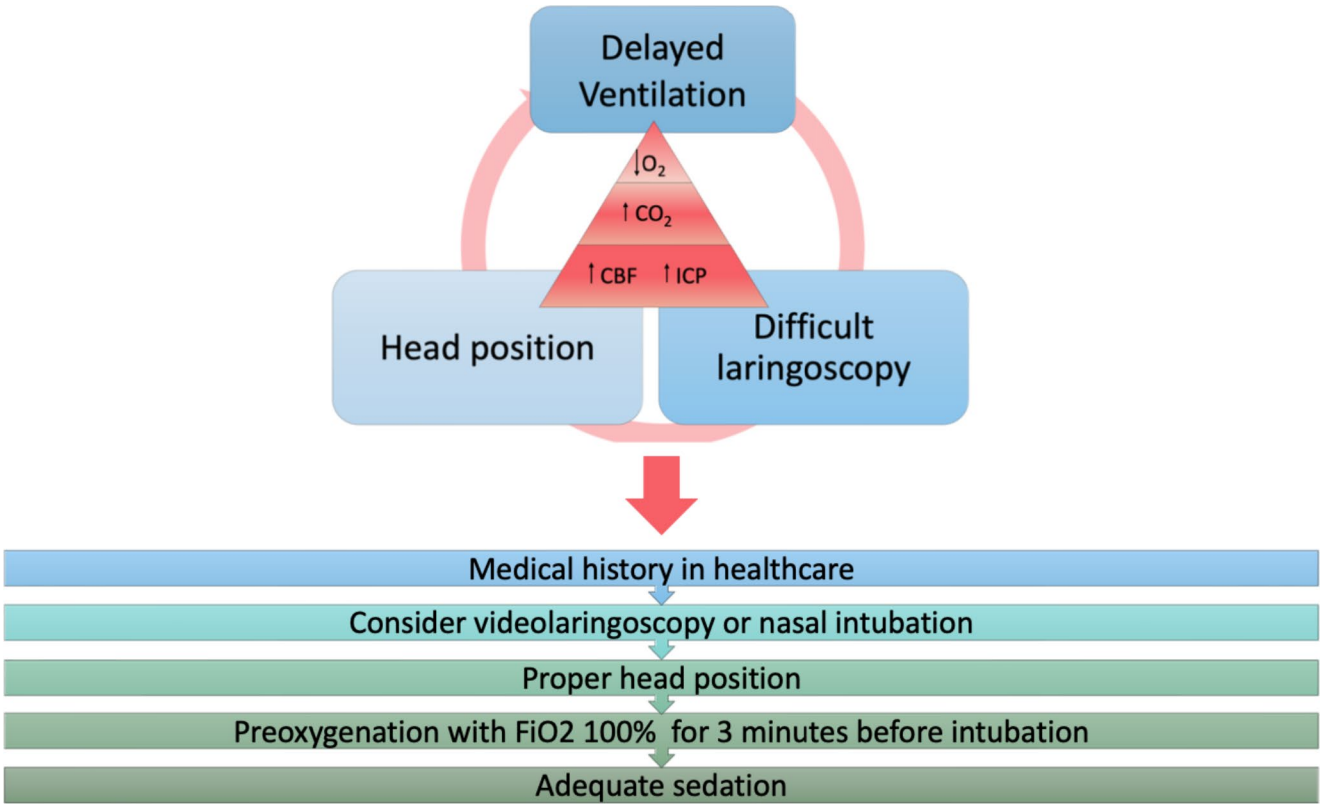

Fig. 1 Airway management in neurosurgical patients

and the brain surgical patients, and intraoperative ventilator settings were not associated with them [39].

Tidal Volume

Specific data on V_T thresholds in neurosurgical patients are limited in the perioperative setting. Knowledge about the impact of V_T on brain is derived from studies conducted especially in critically ill patients with brain injury from ICU cohorts. In a recent meta-analysis conducted in 5639 ventilated patients with ABI, no differences in mortality were shown in patients ventilated with a low V_T strategy (< 8 ml/kg PBW) compared to those who received high V_T (≥ 8 ml/kg PBW) [OR 0.88 (95% CI 0.74 to 1.05), $p = 0.16$, I2 = 20%] [40], but a major risk of PPCs was shown in patients exposed to high V_T (V_T > 10 mL/Kg of PBW) [41].

A consequential risk of low V_T is the potential hypercapnia effect which could be deleterious on cerebral hemodynamics. As known, due to the linear relation between $PaCO_2$ and CBF [42] and because of the perivascular acidosis, cerebrovascular vasodilation and consequent intracranial hypertension can occur in case of elevated CO_2. Given the lack of specific recommendations, and unless low V_T might be related to hypercapnia current knowledge assess the feasibility of LPV in neurosurgical patients [43] applying low V_T (6–8 ml/kg of PBW) [44–47], ideally adjusting it to lung size [43] with the aim to reduce PPCs, [9] minimizing alveolar overdistention and rupture, and lung injury associated to both systemic and alveolar inflammation and setting it to achieve normal $PaCO_2$ levels [42, 48].

PEEP and RMs

PEEP titration can prevent alveolar de-recruitment, reduce atelectasis and intrapulmonary shunt, and thereby improve oxygenation and lung mechanics [49]. The application of recruitment maneuvers (RMs) is still controversial in neurosurgical patients because of concerns for increased ICP, especially in patients with impaired CA [50]. In fact, the risk of intracranial hypertension due to the increase in intrathoracic pressure and reduced cerebral venous outflow is traditionally considered a concern in this population [9], although recent evidence suggests that PEEP application might not compromise ICP, provided that MAP is preserved [51, 52]. In addition, a decrease in venous return and cardiac output may reduce MAP compromising cerebral perfusion pressure (CPP) with a risk of ischemia [53].

Data about PEEP in neurosurgical patients is controversial. In the past, PEEP was used to prevent and treat venous air embolism (VAE) that can occur during sitting positioning craniotomies [54] because of its effect in increasing right atrial and peripheral venous pressures impeding

the entry of air into the central circulation [54]. However, more recent knowledge do not demonstrated decrease in incidence of VAE in patients ventilated with PEEP. [55] In addition, PEEP seems to be related to an increased risk of paradoxical air embolism in the specific scenario of patients with a probe patent foramen ovale. [56, 57].

Historically, clinicians have been cautious regarding PEEP titration due to concerns of increased intrathoracic pressure and ICP. At present, in neurosurgical patients, while it's important to cautiously focus on the hemodynamic status, respiratory mechanics and lung morphology [58] and monitor ICP in those at risk, PEEP should be used with the same principles as for the general ICU population.

Similarly to PEEP, RMs increase intrathoracic pressure and lead to detrimental effects on ICP, although pressure control recruitment strategy may improve oxygenation, reducing the impairment of ICP [59, 60]. A randomized clinical trial, conducted on neurosurgical patients with the aim to understand feasibility of LPV, found a correlation between RMs and hemodynamic adverse events (i.e. hypotension) [43].

In short, high PEEP and RMs are generally avoided and used only in cases of severe hypoxemia under strict neuromonitoring control [36]. In addition, optimization of intravascular volume status before anesthesia induction should be considered to keep hemodynamics stable [61].

Plateau and Driving Pressure

Plateau pressure (Pplat), which reflects the pressure applied at the end of inspiration to small airways, and driving pressure (ΔP) defined as the difference between Pplat and PEEP, can lead to VILI [62, 63] A secondary analysis of five randomized controlled trials (RCTs) and a post hoc analysis of a large multicenter observational study conducted in acute respiratory distress syndrome (ARDS) patients found a clear correlation between Pplat on the first day of MV and mortality [64, 65]. The ARDSnet trial conducted on ARDS patients found that the risk of mortality was lower (31% vs. 39.8%) in patients treated with lower V_T (6 ml/kg PBW) and lower Pplat (< 30 cmH20) compared to those treated with V_T = 12 ml/kg PBW [66]. An association of ΔP with ARDS was found in brain injured subjects [67, 68].

Current guidelines recommend that protective MV should include low Pplat with a threshold value below 20 cmH_20 [43] with an upper limit to 30 cmH_2O in case of concomitant ARDS [2, 45–47] and low ΔP (< 14 cmH20), especially in case of coexistent ARDS and TBI diseases in order to prevent lung damage [68]. The potential caveats for patients with brain injury could be that the gain of a Pplat reduction is often reached by reducing V_T, which may lead to hypercapnia and its potential harmful effects already described [69], therefore a balanced and tailored made approach is always recommended.

Mechanical Power

Mechanical power (MP) is the total amount of energy transmitted by the ventilator to the respiratory system during each breath cycle. Observational data suggest that MP may be a potential mediator of VILI during invasive MV in ARDS patients [70]. Strong associations between MP and outcomes have been described in various categories of invasive ventilated ICU patients [71, 72], and even short periods with high MP are associated with worse outcomes [73]. However, it is currently unclear whether these findings can be extrapolated to patients undergoing intraoperative ventilation, and whether similar associations exist in neurocritical care patients, both in the ICU and in the operating room.

Specific data about MP in neurosurgical patients are not available. A study evaluating the association between MP and PPCs in patients undergoing general anesthesia showed that the exposure to high MP values was associated with increased risks of PPCs (OR 1.34, 95% CI, 1.17 to 1.52); $p < 0.001$) and acute respiratory failure (OR 1.40, 95% CI, 1.21 to 1.61; $P < 0.001$) in the first seven postoperative days [74]. A prospective observational study assessing the MP effects in the intraoperative setting reported that high MP (12.9 ± 4.5 J/min) is a risk factor for developing PPCs suggesting a 12 J/min as a MP cut-off to prevent PPCs [75]. Specific data on neuro patients derived from ICU neurocritical population. Jiang et al. found that high MP enhances the risk of hospital mortality, prolonged ICU stay, and decreased the number of ventilator-free days [76]. Accordingly a secondary analysis of 'extubation strategies in neuro-intensive care unit patients and associations with outcome' (ENIO) study including 1,217 ABI patients who required MV found that the exposure to high MP is associated with poor clinical outcomes (need for reintubation, tracheostomy placement, and ARDS development) [67].

Additionally, it remains unclear whether the association between low MP and improved outcome is due to the fact that less sick patients inherently less often have injured lungs, or have less injured lungs per se. Herein, it is important to note that MP is often low in neurocritical care patients since their lungs are usually uninjured [77]. A relationship of high MP with mortality is reported both in ARDS [78] and non-ARDS patients [79], a value of MP < 17 J/min, associated with appropriate gas exchange and hemodynamics values [80], should be aimed. From the data reported in the LAS VEGAS post hoc analysis in neurosurgical patients, MP had median value of 6.2 J/min [39], suggesting a protective approach generally applied in this population.

The main and easiest way to reduce MP is by reducing V_T [81] However, respiratory rate also plays a role in MP, and if reducing V_T leads to a significant increase in respiratory rate, there is a chance that MP may actually increase. Furthermore, in neurocritical care, it is uncertain whether $PaCO_2$ levels may be allowed to rise to reduce MP, or rather, what level of $PaCO_2$ can be accepted to protect both the brain and the lungs as much as possible.

Gas Exchange

Oxygen and carbon dioxide levels modulation is a chief priority to prevent secondary brain injury [14]. Based on current knowledge, exact thresholds of gas exchange values in neurosurgical patients remain controversial but the recommended target range in traumatic brain injured patients seem to be 80–120 mmHg of PaO_2 independently of ICP elevation, and 35–45 mmHg of $PaCO_2$ in case of no ICP elevation [82]. In general, normoxemia and normocapnia should be aimed, based on the individualization of PaO_2 and $PaCO_2$ values on specific patient and disease.

Hypoxemia and hyperoxemia may both be physiologically detrimental. Current knowledge correlate hypoxemia to precipitating ischemia and risk of brain injury and hyperoxemia to the potential oxidative damage due to the activation of reactive oxygen species (ROS) [83], but data are actually mixed. A study reported that high FiO_2 may be used in patients with secondary brain injury with no negative effects and without an increase in inflammatory markers and incidence of pulmonary complications [84], while other studies showed an increased risk of death in case of elevated PaO_2 [85]. In this sense, oxygenation should be strictly monitored, and a peripheral oxygen saturation (SpO_2) of > 94% should be the target [86, 87].

$PaCO_2$ is a key mediator of CBF [42]. Cerebrovascular vasodilation and consequent intracranial hypertension can occur in case of elevated CO_2; this effect is mediated by a reduction in the perivascular pH. Conversely, a low $PaCO_2$ value can result in cerebral vasoconstriction via perivascular alkalosis, potentially leading to brain tissue hypoxia and cerebral hypoperfusion [88]. Hypocapnia is associated with brain neuronal damage and apoptosis [89]; extreme hypercapnia should also be prevented [88]. A randomized clinical trial of intraoperative LPV in patients undergoing neurosurgical procedures conducted in 60 patients equally divided into control treatment (treated with V_T = 10 ml/kg of PBW, RR at 6–8 breaths/min, no PEEP and no RM) and LPV strategy group (treated with $V_T \leq 6$ ml/kg of PBW, Respiratory rate at 16 breaths/min, PEEP at 5 cmH_20, RM routinely applied after intubation) found higher values of $PaCO_2$ during LPV (mean $PaCO_2$ 35.5 mmHg in control group vs. 37.1 mmHg in LPV group), though the difference was not clinically relevant ($p = 0.002$) [43]. This result should be confirmed by larger studies but suggest again that a personalized approach of MV parameters on the single patient should be recommended.

A recent secondary analysis of the ENIO study suggests that $PaCO_2$ had a U-shaped relationship with $PaCO_2$, where extreme values of hypocapnia (< 25 mmHg) and hypercapnia (> 45 mmHg) were both associated with in-hospital mortality in acute brain injured patients in ICU, suggesting against extreme $PaCO_2$ values [90]. In addition, normal values of CO_2 are associated with decreased mortality and could improve outcome in TBI patients [91]. Probably, because of a concern for potential increased ICP, traditionally, TBI patients were often hyperventilated [92]. This was confirmed in the post hoc analysis of the LAS VEGAS study where lower levels of $ETCO_2$ (32 mmHg vs. 33 mmHg) were found in patients undergoing brain surgery compared to those undergoing spinal surgery [39]. Lower values of CO_2 can be targeted to manage intracranial hypertension, but therapeutic hyperventilation may be considered as an option in brain herniation as a rescue therapy for brief periods (15–30 min), while definitive treatments for ICP management are arranged (e.g. urgent surgery) [93]. Current knowledge suggests to absolutely avoid $PaCO_2 < 25$ mmHg because of the potential risk of vasoconstriction, reduced CBF and increased risk of cerebral ischemia. In absence of herniation risk, $PaCO_2$ should be maintained between 35 and 38 mmHg or 32–35 mmHg in case of refractory intracranial hypertension [42].

Fig. 2 summarizes the issues and MV parameters that could be used in neurosurgical patients.

Ongoing Trials of MV in Neurosurgical Patients

A recent study relating mechanical ventilation during neurosurgical patients (NCT02386683) with the purpose to explore the effectiveness of LPV during general anesthesia for neurosurgical procedures on postoperative pulmonary outcome, compared with traditional ventilation has been concluded but results are still not published. Further studies are required to understand the better intraoperative ventilation strategy to apply in neurosurgical patients.

Management of Intraoperative Conditions Impacting Mechanical Ventilation

Various conditions related to neurosurgical disease can impact MV management, including intraoperative seizures and ICP crises, VAE.

During awake surgery, in case of seizures and status epilepticus, for instance, cold water or cold artificial CSF

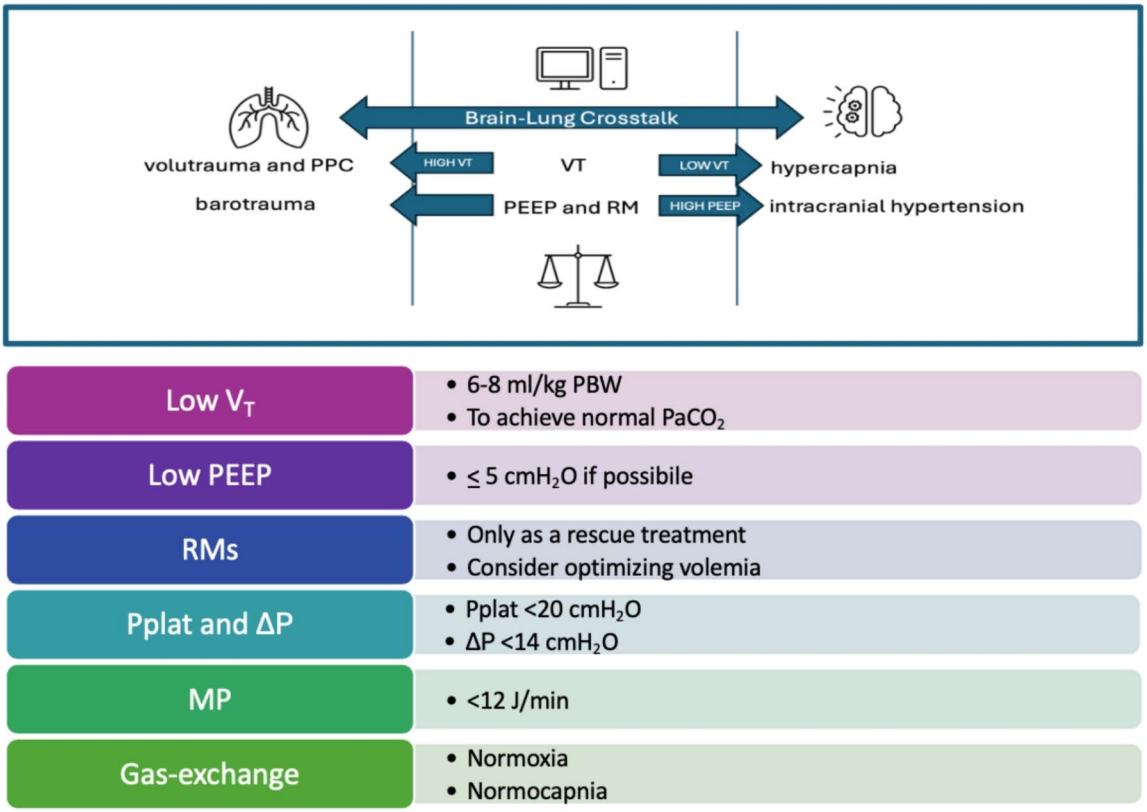

Fig. 2 Mechanical ventilation parameters in neurosurgical patients

can be used on the brain surface to terminate seizures [94]. However, in case of continuous seizures, a switch to general anesthesia with airway protection is mandatory [94].

In patients under general anesthesia, the recognition of seizures could be difficult, and tachycardia and hypertension as a result of sympathetic discharge may be the only signs. [21].

ICP rises need to be prevented and aggressively managed. Appropriate sedation and muscles relaxation are mandatory under anesthesia in order to control ICP by reducing metabolic demand, ventilator asynchrony, venous congestion, and the sympathetic responses [21].

Strict intraoperative monitoring of ICP or of indirect signs, such as pupil alterations and cardiac arrhythmias is warranted [95], aiming at the maintenance of cerebral perfusion pressure and implementation of brain protective measures to decrease $CMRO_2$ and oxygen need. $ETCO_2$ monitoring could help in preventing additional increases in ICP due to hypercarbia [21].

Moreover, VAE may occur during neurosurgical procedures when there is an open connection between the environment and a venous vessel with sub atmospheric pressure, especially those that require a sitting or semi-sitting position (i.e. fossa posterior craniotomies) although this risk was also identified during lumbar laminectomy in the prone position [96, 97]. Saturation, hypotension [98], dyspnea, chest pain and nausea as a sudden drop in the arterial blood pressure and in the $ETCO_2$ value are highly representative signs of VAE, suggesting $ETCO_2$ monitoring as a useful monitoring to prevent this condition [99].

Extubation and Postoperative Pulmonary Complications

Important considerations in determining extubation readiness after neurosurgical procedures include the potential for upper airway obstruction related to different conditions that may develop during surgery. Patients who underwent cervical spine surgery, major head and neck surgery obesity and obstructive sleep apnea syndrome (OSAS), among others, have a significantly higher risk of extubation failure [100].

Risk factors for airway obstruction include prolonged operative time (> 5 h), blood loss > 300 mL, manipulation above the C4 vertebra, myelopathy, operations with > 3 vertebral bodies involved, and use of an anterior-posterior

approach [101]. In addition, pharyngeal stenosis leading to airway obstruction after extubation has been described following an anterior approach in cervical spine surgery involving C0-C2 because of the paralysis of the recurrent laryngeal nerve and overflexion during cervical fixation, causing the protrusion of C2 vertebral body into the pharyngeal wall [102–104]. Similarly, laryngeal edema is possible after spinal surgeries in the prone position [105].

Mainly in neurosurgical patients, during the awakening and extubation phase, it is important to avoid hypertension and coughing to reduce the risk of bleeding and CSF leak [106]. Current knowledge does not provide a right timing or a procedure for performing extubation in neurosurgical patients, but it is undeniable that early and safe extubation might reduce PPCs, diminishing the duration of MV experienced by patients.

In neurosurgery the occurrence of PPCs is closely related with neurological deficits and has a significant impact on patients' quality of life [107–109]. Intraoperative MV plays an important role in the development of PPCs, such as atelectasis, pneumonia, and infections [88]. Neurosurgical procedures are recognized as high risk for the occurrence of PPCs [110].

PPCs adversely influence morbidity and mortality [111], especially within the first postoperative week [112].

In patients undergoing neurosurgical procedures, PPCs are estimated to develop in 13% of subjects, significantly affecting clinical outcomes [34, 39, 43, 113].

Neurosurgical patients who undergo craniotomy [114] as well as patients undergoing posterior fossa surgery are at higher risk of developing PPCs because of the potential for mechanical obstruction, central respiratory dysfunction, and neuromuscular dysfunction [115]. In addition, OSAS is a frequently existing condition in posterior fossa lesions [116]. A recent study analyzed the incidence of PPCs in patients underwent craniotomy for posterior fossa tumors, finding that the most important predictors of PPCs were postoperative blood transfusion, prolonged ICU stay, and tracheostomy and relating PPCs to a longer ICU and hospital stay, and increased mortality [117].

In this sense, LPV strategies including the use of a low V_T, low PEEP and low Pplat seem to reduce the risk of PPCs [2], but its application in neurocritical patients is still debated. A recent randomized controlled trial (RCT) found LPV feasible for neurosurgical patients, reporting no differences in intraoperative adverse events (cerebral tension and $PaCO_2$) between the group treated with LPV strategy and the control group [43].

A post-hoc analysis of the LAS VEGAS study focused on neurosurgical patients undergoing brain or spine surgery showed no differences in PPCs incidence in the spine and the brain surgical groups, and no association between intraoperative ventilator settings and PPCs [39].

Conclusion

Many questions regarding MV in neurosurgical patients remain unsolved and are still under debate.

Neurosurgical patients pose challenges for perioperative anesthesiologist management. The main challenges include the need to use protective ventilation to prevent lung injury while avoiding secondary brain damage.

Firstly, the airway management could be more difficult in these patients. Some may require cervical immobilization, hindering standard extension procedure during laryngoscopy. The fragile cerebral dynamics of this population could be affected by unnatural head positioning, potentially impairing cerebral venous outflow and increasing the risk of ischemia. The risk of hypercapnia, related to a delayed ventilation or a low V_T LPV could lead to increased CBF with an increasing the risk of high ICP, which is dangerous for the patients' dynamics and the risk of bleeding. LPV, generally recommended in abdominal surgical patients for its protective effect on PPCs, may be applied in neurosurgical patients with special attention. Permissive hypercapnia and higher level of PEEP could have detrimental effects in this population. Strict monitoring is needed intraoperatively to prevent eventual intraoperative conditions that can impact MV, seizures or ICP crises.

Much of the knowledge in this area is derived from neurocritical ICU trials or from studies conducted on general surgical patients. A strong statement on more evidence and specific to neurosurgical patients is needed to understand specific ventilatory threshold and risks in this population, possibly distinguishing between brain surgical and spine surgical patients. While waiting for new results, as there is currently no definitive evidence on the feasibility of utilizing LPV strategies in cranial and spinal surgery, we underscore the importance of using a protective MV strategy to reduce the risk of secondary brain injury. This involves titrating MV parameters to the specific patient's need and disease characteristics, with strict monitoring.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Human and Animal Rights All reported studies with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its institutional research committee standards, and international guidelines).

Competing Interests The authors declare no competing interests.

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